ATTACHMENT B

REMARKS

By the present amendment, minor amendments have been made in accordance with the suggestions of the Examiner and to place the claims in more acceptable form. In particular, claim 30 now refers to an isolated antibody that binds to a polypeptide of an amino acid having the sequence of SEQ ID NO: 13 or a fusion protein comprising that sequence. An additional dependent claim has been added which merely reflects that the polypeptide can be coded by the nucleotide sequence of SEQ ID NO: 12 which codes for SEQ ID NO: 13. In light of the amendments and arguments set forth herein, it is submitted that the present application overcomes the prior objections and has been placed in condition for allowance.

In the Official Action, the Examiner maintained the rejection of Claim 30 under 35 U.S.C. § 102(b) as being anticipated by the Palker et al. 1988 PNAS journal reference as evidenced by McGuinness et al. 1993 article in Mol. Microbiol. The Palker et al. article cited by the Examiner relates to synthetic peptides from HIV viral proteins and is thus totally unrelated to the present invention which is directed to antibodies that bind to a polypeptide from *Staphylococcus epidermidis,* a bacterium. The asserted position of the Examiner appears to be that the Palker article discloses the generation of antibodies from peptides which have at most three amino acids in common, and that the McGuinness et al. article shows that NNT is an epitope that can be recognized with regard to a meningococcal strain, a bacteria that is different than *S. epidermidis.* This rejection, insofar as applied to the claims as amended, is respectfully traversed for the reasons as discussed below.

In the first place, as indicated above, the Palker et al. article simply does not disclose or suggest antibodies to the "fig" protein of *S. epidermidis* which is identified as SEQ ID NO: 13. This article relates solely to the generation of a variety of peptides from HIV proteins, a few of which contain the sequence contain the three-amino acid sequence NNT (Asn-Asn-Thr) in the middle of larger peptides, including SP-10 (20 amino acids), SP-11 (23 amino acids) and SP-14 (29 amino acids). Indeed, the sequences around the NNT 3-amino acids location differs greatly than the sequences of the present fig protein of SEQ ID NO: 13. Accordingly, there is no evidence that the NNT region is somehow responsible for generating antibodies in Palker, much less any evidence that any such antibodies in fact recognize and bind to the NNT region of those peptides. As such, there is clearly no indication whatsoever that any antibodies generated as disclosed in Palker would recognize the polypeptide of SEQ ID NO:13, and indeed the fact that these antibodies were generated from viral peptides indicates that they will be unable to recognize a vastly different bacterial protein such as the fig protein of the present application.

Moreover, even assuming that one or more of the viral peptide antibodies of the Palker reference do actually recognize the NNT peptide, such a fact would be irrelevant to the question of whether such an antibody would recognize this extremely small epitope on every other protein simply because a protein contains NNT. Indeed, the McGuinness article also suggests other trimer epitopes which are likely found in thousands of other proteins, but certainly this would not mean that the antibodies generated in Palker can recognize and bind to thousands of proteins of any origin. Even the authors of the Palker article do not disclose or suggest that this is the case. To the contrary, the mere fact that an antibody may recognize a very small epitope such as

NNT clearly does **not** mean that they will bind to every other protein that may contain NNT (or other trimer epitope) because (1) that NNT epitope could be buried in the structure of other proteins (i.e., not surface accessible to antibodies), (2) the NNT might not be immunogenic in every protein, and (3), depending on how the protein folds, the NNT epitope could be sterically protected by other amino acids.

Accordingly, it is clear that the Palker et al. article relating to viral peptides is unrelated to the present claims directed to an antibody that binds to the fig protein (SEQ ID NO: 13) of the bacterium *S. epidermidis* and thus clearly does not disclose or suggest the present claims. The Examiner's rejection of the claims on the basis of the Palker et al. article is thus respectfully traversed and should be withdrawn.

In the Official Action, the Examiner rejected claim 30 under 35 U.S.C. § 102(b) as being anticipated by the Espersen et al. 1990 APMIS article as evidenced by Pei et al. 1999 and Nilsson et al. 1998, neither of these two latter articles being cited as prior art against the present application. The Examiner apparently recognized that Espersen makes no mention whatsoever of the specific fig protein of the present claims, much less any specific antibodies that bind to this polypeptide, but instead argued that this reference inherently discloses the present claims because certain antibodies such as those included in pooled human serum, were able to block the adherence of silicone-catheter binding staphylococci, and that since the pooled antibodies blocked adherence to fibrinogen, these antibodies must inherently be directed to SEQ ID NO: 13. This rejection, insofar as applied to the claims as amended, is respectfully traversed for the reasons as discussed below.

Contrary to the Examiner's assertions, the cited Espersen article contains no disclosure or suggestion of the present claims, namely an isolated antibody that binds to

the fig protein or SEQ ID NO: 13. In particular, the Espersen et al. authors indicated that the main point of the article was that *S. epidermidis* from catheter-related infections adhered no differently than *S. epidermidis* from other sources. For example, in Table 3, page 475, Espersen showed that plasma, serum, albumin, IgG, fibrinogen, fibronectin, and a variety of other materials inhibited the attachment of *S. epidermidis* to silicon catheters. Thus, contrary to showing somehow that the fig protein was somehow isolated and antibodies were directed specifically to this protein, these data evidence a lack of specificity and **not** a fig antibody driven effect. Moreover, the article also discloses that the administration of fibronectin, which is found in serum and plasma at elevated levels, also inhibited adherence, so indeed the article evidences that it was fibronectin, and not any antibodies at all, that was the inhibiting component responsible for the inhibition of attachment shown in the article, as was discussed in the conclusions on page 476. Finally, the Examiner seems to believe that there is only one fibrinogen binding protein in staphylococcal bacteria, i.e., the fig protein, when indeed there are multiple fibrinogen binding proteins in *S. epidermidis* and other staphylococci, and in fact many of these binding proteins generate antibodies that do **not** recognize other fibrinogen binding proteins.

It is thus clear that the Espersen article does not directly or inherently disclose or suggest the specific antibody of the present claims which binds to the fig protein (SEQ ID NO: 13), and thus the Examiner's rejection on the basis of this reference is respectfully traversed.

In light of the amendments and arguments as set forth above, Applicants respectfully submit the present application overcomes all prior rejections and has been placed in condition for allowance. Such action is earnestly solicited.

END OF REMARKS